NASA TTF-9497

CEREBRAL RHEOGRAPHY AND ITS DIAGNOSTIC POSSIBILITIES IN CLINICAL PRACTICE

NASA TTF-9497

A. Belluschi and V. Vacchini

Translation of "La Reografia Cerebrale Sue Possibilita' Diagnostiche Nella Pratica Clinica".
L'ospedale Maggiore, Milano, Vol. 59, pp. 201-238, 1964.

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON D. C. AUGUST 1965

NASA TTF-9497

CEREBRAL RHEOGRAPHY AND ITS DIAGNOSTIC POSSIBILITIES IN CLINICAL PRACTICE*

A. Belluschi and V. Vacchini

ABSTRACT 33261

The authors have taken up the problem of interpreting cerebral rheography. After having reported upon present knowledge on the subject, they examine the diagnostic possibilities of this knowledge and come to the conclusion that the interpretative procedure is at present entirely inadequate for furnishing data worthy of attention.

Basing themselves on the hemodynamic laws which define the behavior of the cerebral flow and taking advantage of the ideas expressed in various authors' investigations using the nitrogen protoxide method, they see cerebral rheogram interpretation prospectively as an expression of the flow as a function of vascular resistance and arterial pressure.

To confirm this interpretative procedure, they give numerous examples from their case histories showing an effective correspondence between the ascending limb of the rheographic wave and the vascular resistance

Author

* Conference held on March 4 in the Physiotherapy Lecture Room of the Ospedale Maggiore.

and area under the curve as a function of the relationship between pressure and vascular resistance.

Interest in measuring the volumetric variations of various regions of the body by using impedance changes has become widespread among physiologists and clinicians since the early nineteen-hundreds, but only in recent years with Kolber, Polzer, Marko, Piaher, and Schuhfried (1949-1955) have we had at our disposal equipment permitting the introduction of this type of measurement into clinical practice. /201*

Owing to the advance of electronics, we now have an apparatus which is easy to use and faithful in its reproduction of electrical phenomena, but the problem arises of interpreting them in order to be able to offer the medical faculty a new investigatory procedure. The rheogram represents a recording of the impedance changes in a definite region of the body; these changes are linked to changes in electrical conductivity, which in turn is a function of density and volume.

Since the blood is the only element offering quantitative fluctuations in a given region, the rheogram which we record must be the expression of the rhythmic fluctuations in the influx of blood in the region in question.

THE RHEOGRAPHIC TRACING

Morphological Elements of the Rheographic Tracing

The rheographic tracing evinces morphological similarities to the sphygmogram, oscillogram, and ECG. It is composed of a wave comprised /202

* Note: Numbers in the margin indicate pagination in the original foreign text.

of an ascending link and a descending link united at a point called the peak. Since an increase in electrical conductivity is indicated by an elevation of the isoelectric line, and since diminution depresses this line, this morphology expresses the rhythmic afflux and efflux of the blood - the rising limb of the wave corresponding to afflux, and the falling limb corresponding to efflux.

Since blood inflow and outflow, in turn, are a function of the vascular resistances of the elasticity and tone of the vessel walls, it is assumed that the rheographic tracing expresses the result of all these interrelated factors. The search for a correlation between these factors and the individual limbs of the rheographic tracing has become the object of much research, whose results are still differently interpreted and discussed. Let us relate in detail the morphological elements which are generally offered to interpret a peripheral rheogram (Figure 1).

a - Deflection or rising rheographic limb: this is due to an increase in electrical conductivity, and is the expression of the arterial systolic afflux. Its amplitude and duration are a function of the magnitude and duration of the afflux of blood. Moreover, it has been extablished that the ascending limb of the rheogram is more or less reduced as the diameter of the vessel under examination becomes smaller (Ref. 21-27).

b - The time which passes between cardiac systole and the arterial vasodilatation in a given region. It can be deduced by comparing the q deflection of the ECG. The start of the ascending limb of the

rheogram is called the q-base time, and serves for deducting the velocity of the sphygmic wave.

c - The time which passes between the start of the ascending rheographic limb and its peak determines the duration of the ascending limb and expresses the manner in which the vascular wall expands. This is designated as the rise time.

d - The peak or apex of the rheographic wave is the point of transition between ascending and descending limb, and corresponds to the moment of quantitative reversal of electrical conductivity, and in turn corresponds to the crest of arterial afflux and beginning of efflux.

e - The deflection or descending rheographic limb signifies the reduction of electrical conductivity and corresponds to a volumetric decrease resulting from the efflux of blood.

f - A constant dip of the descending rheographic wave is the /203 dichrotic wave which intercalates itself between the middle third and the upper third; this is associated with vascal elasticity, but its true significance is at present moot.

g - A secondary dip is now and then observed in a few patients at the base of the curve, and is considered to be synchronized with the atrial activity.

h - Total time of rheographic wave. This is the time which elapses between the base and the end of the descending limb and indicates the period of the entire systolic-diastolic cycle. This together with the rise time serves to compute sphygmic velocity.

$$i - \frac{\text{Rise time}}{\text{Total duration of rheographic wave}} \times 100 = \text{Sphygmic velocity.}$$

This functional index is of the greatest importance insofar as it informs us - with the exclusion of the cardiac causes affecting the duration of the ventricular expulsion period - of the possibilities of the more or less rapid dilation of the vessel walls under the effect of the sphygmic wave, thus furnishing data which reflect the elasticity and the tonus of the vessel walls themselves. The lower this index is, the more rapid is the rheographic phenomenon.

ℓ - Sphygmoplanimetry. The amplitude of the rheographic tracing, the electrical conditions being equal, is due to diverse factors which vary in other respects from region to region under examination. In general, the factors which modify the amplitude of a rheographic tracing are:

-- endovascular arterial pressure and its variations,

-- slackening, or stasis, of venous circulation,

-- pulse beat,

-- cardiac frequency with relative modifications of the pulse beat,

-- position of the regions of the body,

-- vascular elasticity, and

-- vasomotor tone.

In general, a vasodilatory state increases the sphygmoplanimetric amplitude of the rheogram where the ascending limb becomes more slanting, the peak rounded, and the dicrotic wave less evident. A vasoconstrictive state decreases the sphygmoplanimetric amplitude of the rheogram /204
where the ascending limb becomes steeper, the peak sharper, and the

dicrotic wave more accentuated.

While some authors give more consideration to the amplitude and planimetry of the rheographic curve with their absolute numerical data, other authors prefer to attribute more significance to its morphological characteristics (Ref. 19, 27, 34, 49, 57, 70, 72).

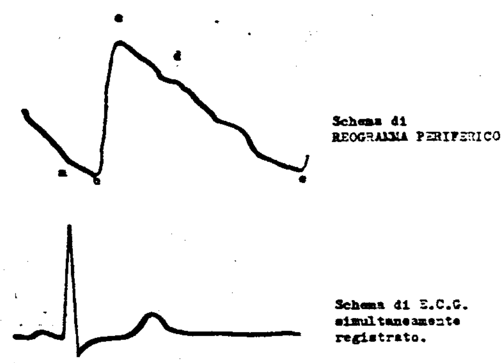

Figure 1

[Top Figure: Diagram of Peripheral Rheogram]

[Bottom Figure: Diagram of Simultaneously-recorded ECG]

a-b = q-base period of rheographic wave
b = base of rheographic wave
c = peak of rheographic wave
b-c = rise time
d = dicrotic notch
b-d = total duration of rheographic wave

Having recounted the elements for interpreting a rheographic tracing, let us now provide a diagrammatic example of a normal peripheral tracing, with the statement that the rheogram nevertheless varies for every individual.

CEREBRAL RHEOGRAPHY

Not to mention the constant electrical conditions (calibrations of equipment and tracing during recording), the ease of recording and the absolute harmlessness of the rheogram have indicated that it would be useful in studying cerebral hemodynamics, which up until the present has confronted investigators with significant technical difficulties. The first scientific contributions on cerebral rheography by Aninger et al., Kainde, Merlen, Doizelot, Polzer-Schuttfriend, Posteli, etc. go back to 1950-1951. Since then, other works have appeared, among which we might mention those of Pratesi, Nuti, Sciagrà, Feruglio, Posteli, /205
Garbini, Montali, Serra, Kunter, etc.

Although by now interpretive elements have been obtained which are both physiologically and pathologically certain, the cerebral rheogram still remains little known and is the property of a limited circle of researchers, particularly because of the difference in its clinical interpretation.

The cerebral rheogram measures the overall impedance variations which are recorded between two electrodes placed in different positions on the head. The impedance variations are generated by flux changes which may be simultaneously attributed to the different systems of the internal and external carotid arteries and the vertebral artery.

Since the endocranial vascular network is by far more voluminous than the esocranial, it is assumed that the rheogram is the predominant expression of variations within the limits of the endocranial region. Moreover, experiments by Kaindll on animals have made it clear that the

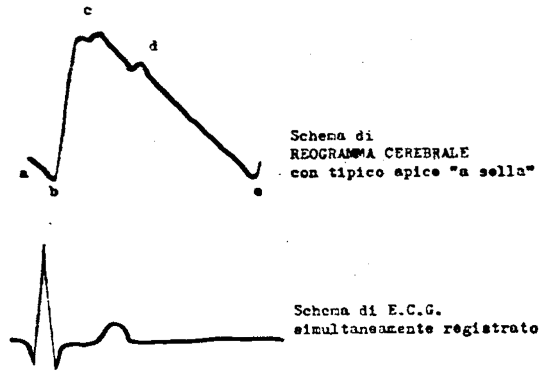

Figure 2

[Top Figure: Diagram of Cerebral Rheogram with Typical "Saddle" Peak]

[Bottom Figure: Diagram of Simultaneously-Recorded ECG]

a-b = q-base period of rheographic wave
b = base of rheographic wave
c = peak of rheographic wave
b-c = rise time
d = dicrotic notch
b-e = total duration of rheographic wave

cerebral rheogram obtained with electrodes placed on the scalp is similar to that obtained with electrodes embedded in the cerebral parenchyma. The cerebral rheogram reflects all the characteristics of the cerebral circulation, in which - in addition to the common factors regulating the circulation of the blood (systemic arterial pressure, heartbeat, vascular resistances) - other proper and specific factors come into play, such as the inextensibility of the cranium, fluid incompressibility, and the peculiar condition of venous pressure which, in contrast to other regions, is favored by the force of gravity in the erect position. These characteristics are reflected in the morphology /206

and other elements of the cerebral rheographic tracing. Let us now consider these characteristics and the meaning which different authors have been able to assign to them (Figure 2).

a - q-base period: records the transmission time of the sphygmic wave from the left ventricle to the carotid and the vertebral arteries; its normal values are about 0.12-0.15 sec.

b - Ascending limb: it is normally steeper. By indicating the electrical conductivity increase due to arterial afflux, this limb expresses - by greater or lesser steepness - the arterial dilation in face of the systolic impulse.

c - Rise time, varying from 0.06 to 0.08 sec according to age and indicating the duration of the ascending stroke, provides a further element for evaluating the manner and speed with which the vessels expand.

d - Peak: it presents a typical saddle deflection in the form of a small secondary wave preceding the dicrotic wave. This "saddle" shape of the peak is specific to cerebral rheographs and seems to be attributable to a damping of the sphygmic wave because of the inextensibility of the skull and the incompressibility of the fluid mass. Some authors say that this is present and clearly evident in all the readings; other authors state that it is more visible in the glabella-occipital reading (Aninger, Douzelot, Merlen).

Since the peak becomes rounded when the rheogram sphygmoplanimetry increases, because of the elevated flow of blood, we infer from this that the peak shape is an indirect index of the duration of maximum

afflux.

e - Descending limb: gently falling, at least in the great majority of cases. It presents a dicrotic wave which is always well-marked and is located between the middle and upper third; this is considered to signify the arterial efflux in the diastolic phase. Since this is conditioned by the drainage of the venous channels, its alterations also reflect possible changes in these channels.

f - Finally, catadicrotic fluctuations in the descending limb are recorded which are generated by particular conditions of venous system repletion. They may appear either in physiological conditions such as inspiration, or in pathological conditions involving difficulty of venous discharge.

g - Sphygmic velocity. This (the percentage ratio between duration of ascending limb and total duration of the sphygmic wave) is an expression of the vessels' capability of distending under the sphygmic impulse. /207

h - Sphygmoplanimetry. The cerebral rheogram at its maximum presents a sphygmic amplitude less than that of the peripheral rheogram. This is due to the peculiar cerebral circulation which is characterized by an anatomically-anastomosed, but functionally-terminal type of vascular network which flows into tissue whose volume does not vary.

Particular importance is laid on the sphygmoplanimetric variations of individual readings (Ref. 19); we shall present the standard physiological values of sphygmoplanimetry in different readings:

fronto-occipital -- between 8-12 mm

bitemporal -- between 8-10 mm 1 mm = 0.02 ohm

glabella-occiput -- between 10-8 mm

The following five readings (Figure 3) examining the area between the two corresponding electrodes are generally used:

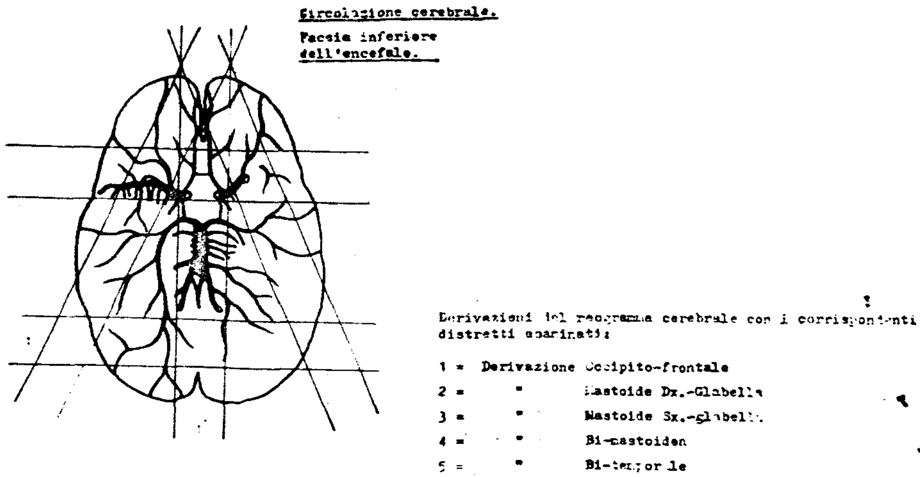

Figure 3

[Top legend: Cerebral circulation. Inferior aspect of brain.]

[Bottom legend: Cerebral Rheogram Readings Examining the following regions:]

1 = Occipito-frontal
2 = Right mastoid-glabella
3 = Left mastoid-glabella
4 = Bimastoid
5 = Bitemporal

Some authors have been engaged in corroborating or disproving /208 the assumptions set forth above in regard to rheographic recording in typical pathological conditions involving cerebral hemodynamics (Ref. 16, 19, 25, 50, 70, 61). Forms of disease have been studied which are characterized by decreased extensibility of the arterial

vasal lumen: arthritis, systemic hypertensive states in renal aclerosis, and essential and arteriosclerotic hypertension. Under these conditions, the cerebral rheogram was taken with the ascending limb typically retarded, the peak a bit rounded, the descending branch very sloping, the dicrotic wave absent or barely accented, and the sphygmic velocity very often increased.

In particular, in case of meningoencephalopathy the cerebral rheogram always proved to be changed. Usually, the change evidenced in the graph reflects some arteriopathy with increased vascular resistances, i.e., it is analogous to the preceding one, even if the genesis of this graph seems to be due at times to changed venous efflux from the endocranial sinuses, at other times to endocranial hypertension, and at still others to vascular compression from meningoencephalic edema, or to these situations in various combinations.

In cases of focussed cerebral vasculopathy, rheographic recording has demonstrated a sphygmoplanimetric difference between the two hemispheres, with reduced amplitude in the hemisphere containing the seat of the lesion.

Successive examinations at differing distances in time from the acute attack have pointed out the evolution of the vasculopathy, with a progressive return of the planimetry to normal or to a more or less complete hyposphygmia - indicating an anatomic disturbance which has now become irreversible.

Rheographic investigation has not proved capable of informing us as to the importance of the lesion, since the hyposphygmia in the

affected hemisphere is the same in the case of either a mild or an extensive lesion.

Not infrequently an anisosphygmia between the two hemispheres has been found in cerebral rheograms made of subjects without clinical pictures of acute vasculopathic cerebral episodes. This has been interpreted as a subclinical state of focussed vascular affection latently predisposing to possible apoplectic episodes.

Rheographic investigation has frequently thrown light on the functional genesis of an acute vasculopathic episode, when the rheograms recorded at short time intervals have evidenced a clear and quick regression in the sphygmic change.

Rheographic investigation of the individual cerebral hemispheres has been proposed before and after homolateral and contralateral carotid compression, with the possibility of diagnosing the possible presence of any collateral circle through Willis' heptagon and any stenosis, /209
partial or complete, of an internal carotid.

Finally, one localized vascular cerebropathy, which is clearly revealed by cerebral rheographic investigation, has been arteriovenous fistula which had the following peculiar graphic signs: anisosphygmia between the two hemispheres with hyposphygmia of the hemisphere containing the seat of the aneurysm and a normal rheogram of the undamaged hemisphere. The rheogram of the affected side evinced a roundish morphology with remarkable sphygmic amplitude, lack of dicroticism, and absence of other secondary properties.

In idiopathic cephalalgia, rheographic findings have indicated a

vasodilatory or a vasoconstrictive situation. In the first situation, in fact, mild hyperplanimetry has been found with the ascending limb somewhat retarted, a peak with a well-marked saddle, and a barely discernable dicroticism. In the second situation, the tracing presented mild hypoplanimetry, rising limb a bit steep, pointed peak, and marked dicroticism.

In some extracerebral involvements, the cerebral rheogram may undergo modifications in the case of systemic vascular afflictions, as in periarteritis nodosa, generalized arteriosclerosis, Buerger's disease, coarctation of the aorta - where the possible existence of a marked secondary circulation has been indicated with hyperplanimetry in tracings with electrodes placed on the occiput, probably detecting the compensatory circulation carried out by the vertebral artery and its collaterals.

PERSONAL RESEARCH

The bibliographical information shows cerebral rheography to be a method of investigation which is rich in diagnostic potentialities and with numerous applications. Comparing the tracings which various authors have published, we find convincing correlations between the changes in the rheogram and the clinical manifestations pointed out. Extending our observation further to a wide range of cases, we conclude that the changes described are not after all so typical, because in the last analysis the same changes in the individual limbs (to which a definite meaning is attributed) are present in different pathological

situations which do not have the same change in common. It is, however, true that the explanation of the individual tracing may be interpreted on the basis of clinical findings, but the fact that the same change is variously interpreted eliminates the possibility of formulating a diagnosis independently of the clinical finding. To confirm this, in Figures 4, 5, and 6, we present groups of cerebral rheograms of patients with three different pathological conditions - that is, severe anemia, /210
arteriosclerosis, and hypertension. It is evident that the rheographic tracings of every group, although expressing the same pathological condition, differ greatly among themselves, and without the aid of clinical diagnosis it would be difficult to come to interpretative conclusions. But without denying the implicit value of the preceding research of the different authors and without failing to recognize the validity of many findings relative to the morphological interpretation of the rheographic outline, we have, on the basis of our clinical experience, come to the conclusion that the present method of interpretation is entirely inadequate for making a valid contribution to the clinical diagnosis of hemodynamic disturbances in the cerebral region.

It is for this very reason that we again wanted to tackle the problem of interpreting the cerebral rheogram, by tracing the ideas which have been presented in investigations conducted by the Kety and Schmidt method, which serves to determine the cerebral flux and indirectly to evaluate other important functions, such as the behavior of the cerebral vascular resistance and the metabolic consumption of oxygen.

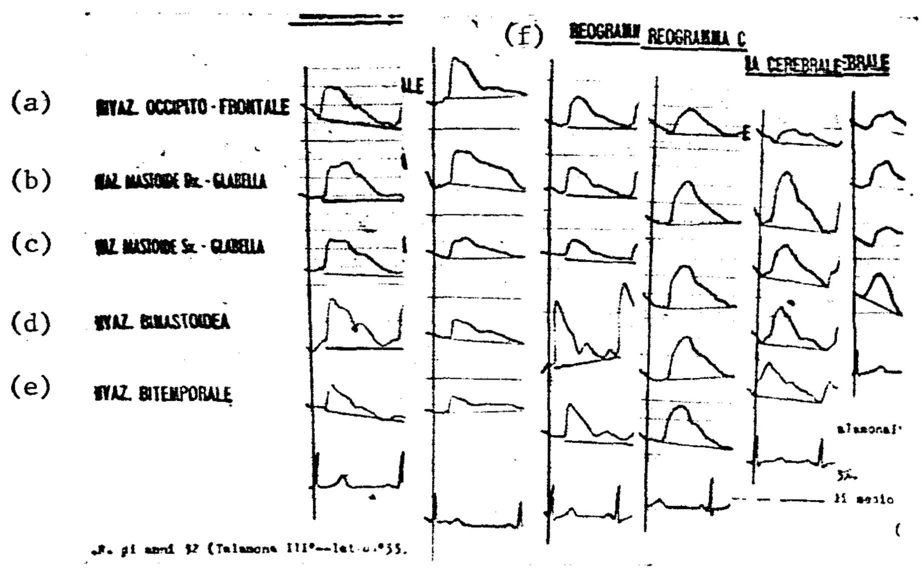

Figure 4

Cerebral Rheograms of Patients with Severe Anemia

(a)- Occipito-frontal reading; (b)- Right mastoid-glabella; (c)- Left mastoid-glabella; (d)- Bimastoid; (e)- Bitemporal; (f)- Cerebral rheogram.

All research performed in the last fifteen years has always /212
taken account of these factors: flow of blood, vascular resistances, arterial pressure, and 0_2 consumption, which are intimately interrelated. These correlations are expressed in the following formulas:

$$\text{Vascular resistances} = \frac{\text{Arterial pressure}}{\text{Vascular resistances}},$$

$$\text{Cerebral blood flow} = \frac{\text{Arterial pressure}}{\text{Cerebral blood flow}},$$

Arterial pressure = Vascular resistances X Cerebral blood flow - from which it follows that if we know two of these factors we can deduce

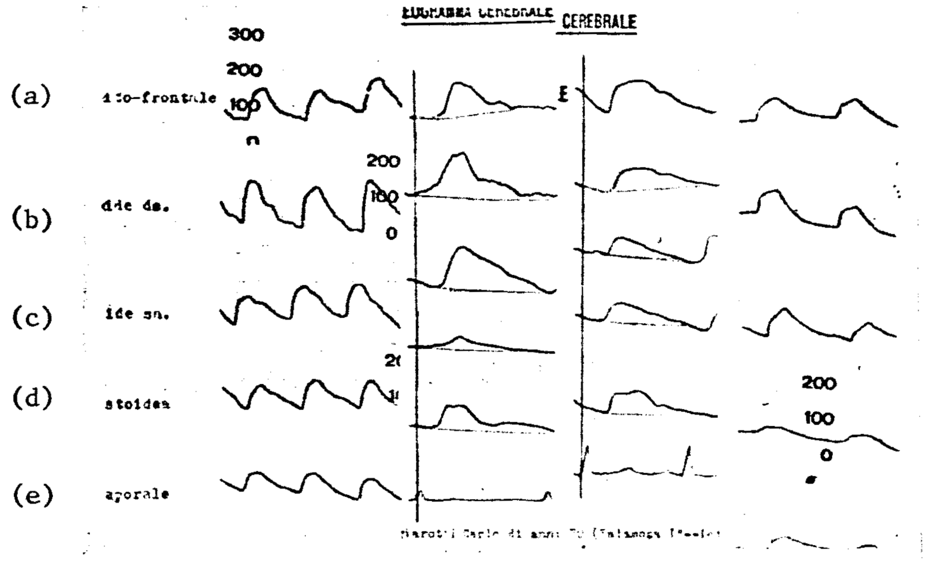

Figure 5

Cerebral Rheograms of Arteriosclerotic Patients /211

(a)- Occipito-frontal reading; (b)-Right mastoid-glabella; (c)- Left mastoid-glabella; (d)- Bimastoid; (e)- Bitemporal.

the behavior of the third.

Since arterial pressure can be easily recorded, we have posed ourselves the problem of whether cerebral blood flow can be deduced when the rheographic outline is able to express the behavior of the vascular resistances.

In other terms, the problem consisted of studying the correlations between rheographic outline and vascular resistance behavior.

If we study the hydrodynamic diagrams, we can anticipate the reciprocal relationships between pressure, flow, and resistances. In Figure 7, we see three containers of equal volume (10 liters) with different-sized spouts (different values of resistance), which are

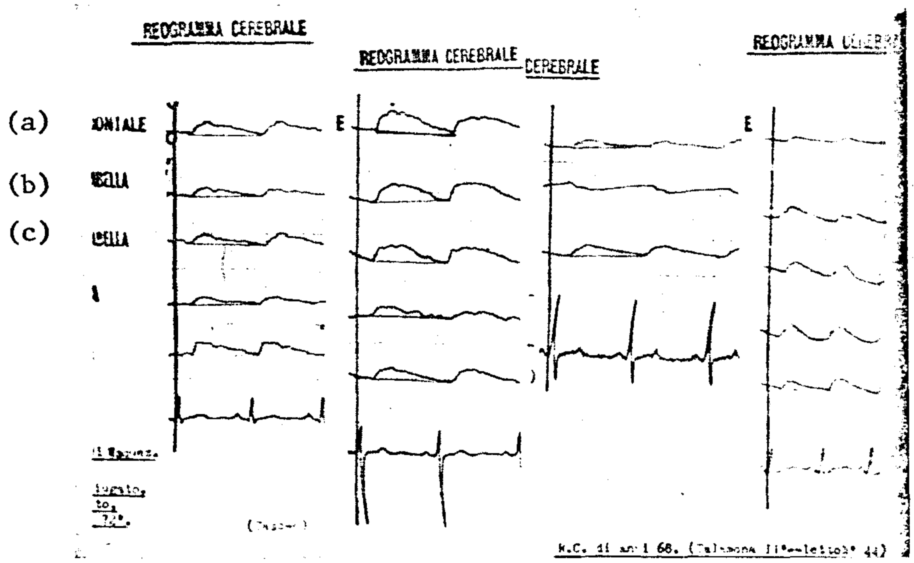

Figure 6

Cerebral Rheograms of Patients with Hypertension
(a)- Occipito-frontal reading; (b)- Right mastoid-glabella; (c)- Left mastoid-glabella.

subjected to different pressures so that they empty in the same amount of time (constant output flow). As is seen, the manner in which the liquid leaves the spouts differs, but the same result is obtained. In the first case, the stream is reduced in size, but - being under greater pressure - it comes out in a longer flow; in the third case, we have the opposite situation with a thicker, but shorter flow. This figure shows how the same flow can take place in different ways. Since the rheographic outline expresses the way in which the flow takes place, it can be seen how different outlines may express the same flow condition. The above examples refer to hydrodynamic aspects in rigid tubes; therefore, they are not entirely applicable to the hemodynamic conditions of the human body which occur in elastic tubes. This is the

reason that the rheographic outline is presented as a typical wave, which, however, within certain limits obeys the same laws - which we can use as examples to which to refer the rheographic outline within the spatial limits of the above examples.

The clinical equivalent of these three examples could be repre- /213
sented by three normal individuals with different arterial pressures and equally different vascular resistance values.

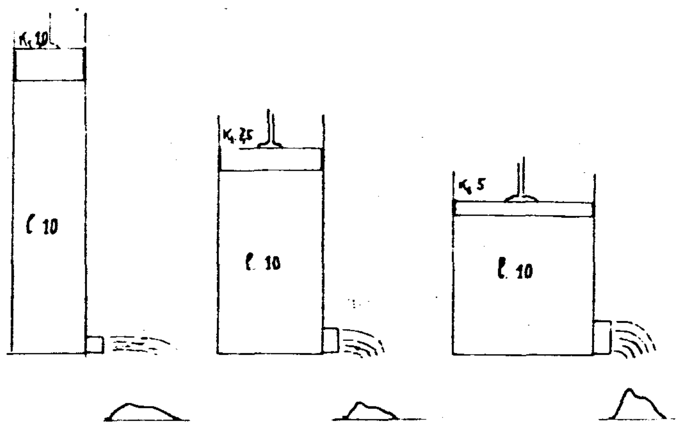

Figure 7

Let us consider in Figure 8 the same conditions of volume and pressure, but with spouts of constantly-reduced size (increased resistances). Since the pressure also varies with the resistance, the output flow varies (the three containers no longer empty in the same amount of time). In the first case, with heightened pressure the flow is still sufficient, but in the third case the flow is decidedly reduced (the stream is scanty and short). The corresponding rheographic

outlines (all three with the ascending limb of the same height, but with progressively-reduced area under the curve) reflect a progressive reduction in flow because of insufficient face pressure and heightened resistances. The clinical equivalent of these situations could be represented by an irreversible hypertensive arteriosclerotic state with progressive cardiac insufficiency.

In Figure 9, we now consider the opposite condition, i.e., a constant pressure with progressively-smaller resistances; it follows from this that the flow increases. The corresponding rheographic outlines show a limb which constantly ascends higher and an ever larger area under the curve. The clinical equivalent could be a hypertensive state not contrabalanced by vasoconstriction, with consequent incipient hemorrhage (aneurysm, plethora, arteriosclerosis of the large vessels).

In Figure 10, we consider, finally, resistance changes unaccompanied by equivalent pressure variations; the flow will vary by lack /214

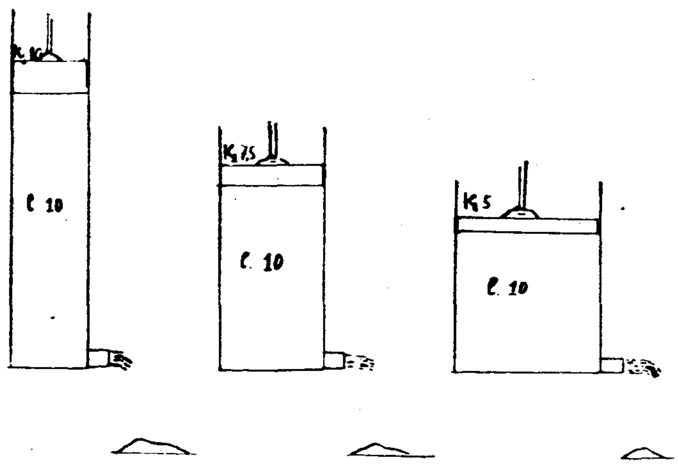

Figure 8

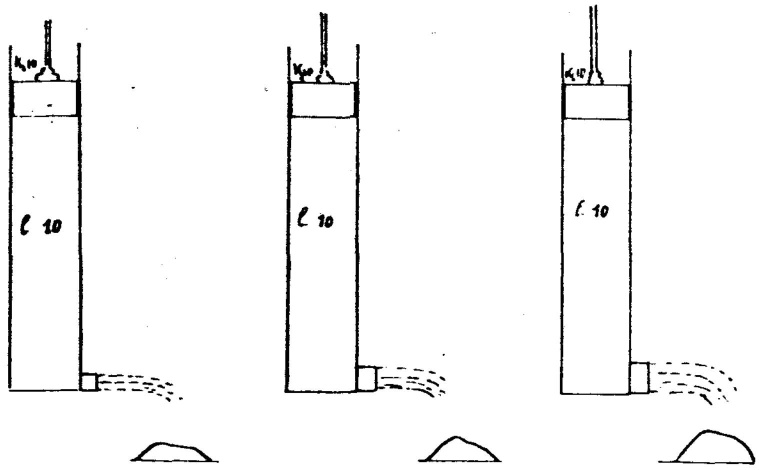

Figure 9

of them in the first case, and by an excess in the third. The rheographic outline presents variations in the ascending limb corresponding to resistance variations, and different areas under the curves equal to the pressures.

The clinical equivalent of these conditions could be represented by regional vasomotor symptoms unaccompanied by variations in general arterial pressure (hyporeactivity of the carotid sinus, vasospasms, thromboses, etc., etc.).

In Figure 11, we present all the above examples for comparative /215 purposes. We can see how the different rheographic profiles at the upper left correspond to a constant flux by means of inverse variation of pressure and resistance. On the contrary, in the other quadrants the various rheographic profiles correspond to different flows by predominant variation of a single one of the two factors, pressure and

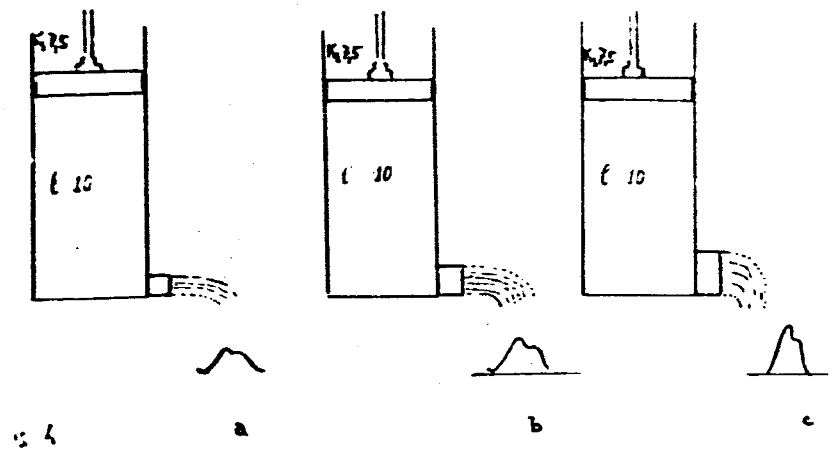

Figure 10

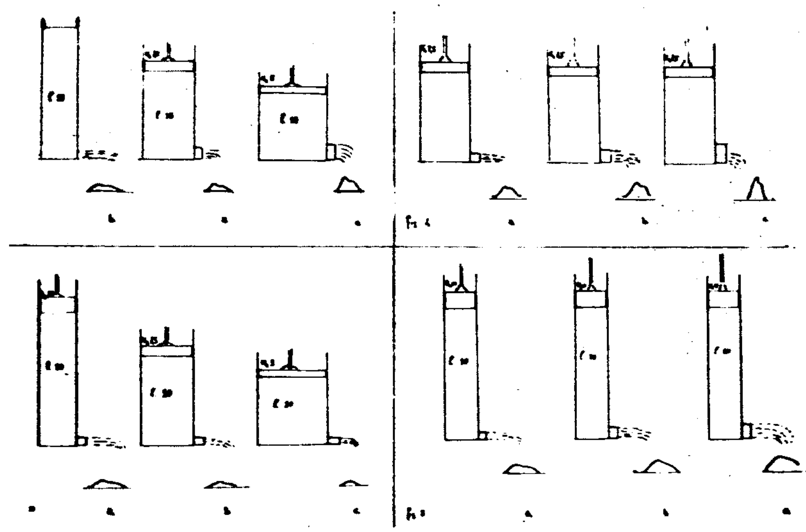

Figure 11

vascular resistance. This figure shows the multiple aspectual variety which the rheographic profile can assume. These rheographic variations /216 and differing rheographic profiles corresponding to diverse flows can be interpreted by a logical criterion if put into relationship with the pressure and resistance variations. Assigning to the height of the

ascending limb the value corresponding to the value of the vascular resistance, and to the length of the rheographic wave - or the total period of this wave - a correspondence with the pressure state, we can arrive at the following figure which represents the possible combinations (Figure 12). We represent the progressive values of arterial pressure as they increase in value on the abscissa, and on the ordinate, the progressive values of the vascular resistance. Every tracing in this case represents the combination of these two different values. The rheographic outlines between the diagonals, while differing in profile, all represent a normal flux; those at the lower left correspond to reduced flow, and those at the upper right to increased flow.

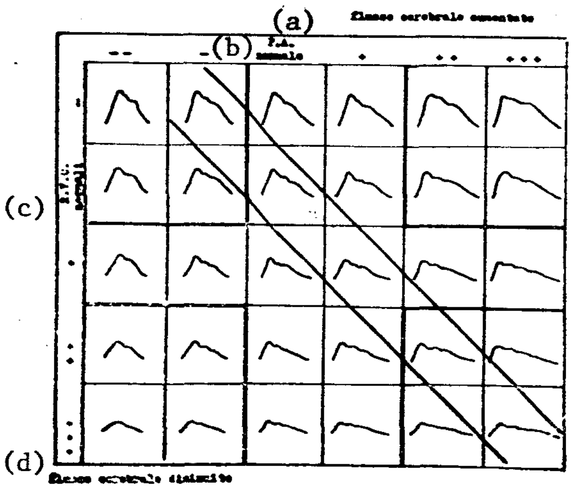

Figure 12

Schematic Representation of Different Cerebral Rheograph Profiles as Functions of Arterial Pressure and Cerebral Vascular Resistance

(a)- Increased cerebral flow; (b)- Normal arterial pressure; (c)- Normal cerebral vascular pressure; (d)- Reduced cerebral flow.

Seen in the perspective of the laws of hydrodynamics, this diagram obviously cannot be adopted in the absolute sense as an appraisal of the cerebral hemodynamic conditions, because of the interference here of many variable factors (whether the vascular resistance value or the value of arterial pressure, they in turn are the result of variable factors). However, it can well illustrate an interpretative procedure based on knowledge of resistance and arterial pressure values.

In order to determine whether this interpretative procedure can find a correspondence in clinical practice, we have collected a set of tracings under experimental and pathological conditions which note the behavior of resistance and cerebral flux which is already the subject of study on the part of numerous authors who have adopted the methods of Kety and Schmidt. We list in the following table (Figure 13) the conditions which are the subject of this research. /217

Methods

The tracings were taken with a Galileo Rheograph under load conditions which were as constant as possible; the arterial pressure was taken simultaneously at the brachial artery on a Riva-Rocci machine. At the same time, all patients underwent examination of the fundus in order to have an indirect idea of the vascular conditions.

Results

The first group of tests concerned the behavior of the cerebral rheogram in young and healthy subjects, subjected to a state of hypercapnia by breathing used air in a closed circuit.

An increase in CO_2 in the blood and alveoli is known to increase

	P. A. [Arterial Pressure]	C.V.R. Cerebral Vascular Resistance	C.B.F. Cerebral Blood Flow in cc per 100 g of Brain	C.M.R.O. Cerebral Consumption of Oxygen
Hypercapnia	+	Reduced	Increased	Unchanged in Tendency
Hypoxemia	+	Reduced	Slightly Increased	Unchanged in Tendency
Arteriosclerosis	Unchanged	Unchanged	Slightly Reduced	Slightly Reduced
Essential Hypertension	Increased ++	Increased ++	Slightly Reduced	Unchanged in Tendency
Arteriosclerotic Hypertension	Increased +++	Increased +++	Reduced ++	Reduced
Anemia	Increased	Reduced	Increased	Unchanged in Tendency
Polyglobulism	Increased	Increased	Reduced	Unchanged in Tendency

Figure 13

the cerebral flow by reducing the vascular resistance value and increasing pressure. The sole aim of both of these reactions is to keep the cerebral consumption of oxygen within normal limits by means of a greater blood /218 flow. The reactions, whose corresponding effect we should look for in the rheogram, are therefore an increase in flow, decrease in vascular resistance, and a moderate increase in arterial pressure.

The tracing in Figure 14 belongs to a 23-year-old student, male, arterial pressure 105/75 mm Hg; the second (Figure 15) belongs to a patient, male, 21, arterial pressure 125/80 mm Hg; and the third (Figure 16), to a subject, 40, arterial pressure 145/90 mm Hg. Examination

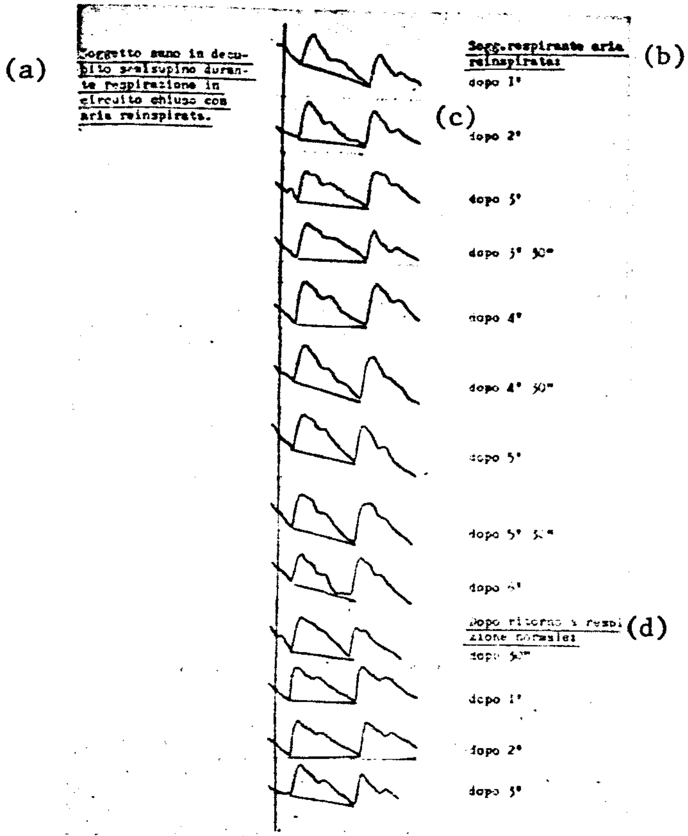

Figure 14

(a)- Healthy subject, semi-reclining, breathing used air in a closed circuit; (b)- Subject breathing used air; (c)- After; (d)- After return to normal respiration.

of the fundus of all three gave findings within normal limits.

The different amplitudes of these three tracings may correspond to the different values of arterial pressure and the relative different values of vascular resistance, in addition to the peculiar individual anatomical characteristics of the skull.

Observing the tracings corresponding to the progressive periods of the experiment, we can see that the rheographic profile becomes /219

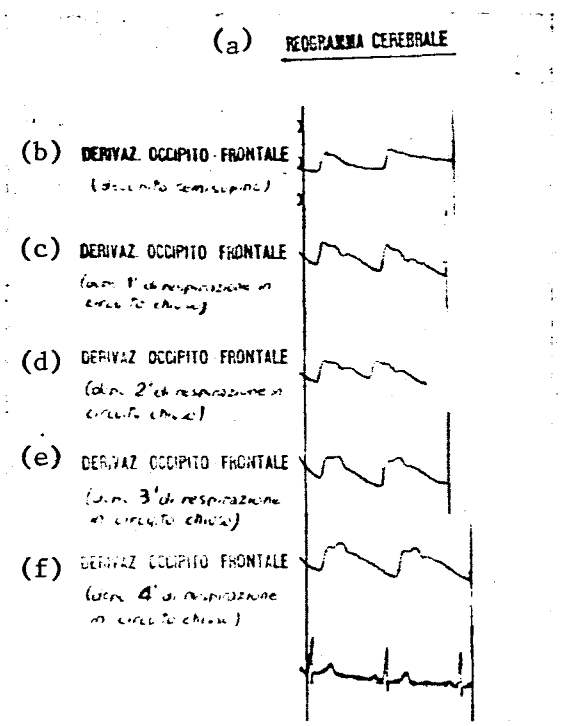

Figure 15

(a)- Cerebral rheogram; (b)- Occipito-frontal reading (semi-reclining); (c)- Occipito-frontal reading (after 1 min. of closed circuit breathing); (d)- Occipito-frontal reading (after 2 min. of closed circuit breathing; (e)- Occipito-frontal reading (after 3 min. of closed circuit breathing); (f)- Occipito-frontal reading (after 4 min. of closed circuit breathing).

modified, tending towards an increase in the area under the curve, which is effectuated by the greater height of the rising limb, broadening of the peak, and less steepness in the descending limb.

We may conclude that in all three subjects, during the state of artificially-induced hypercapnia, the same cerebral rheogram changes

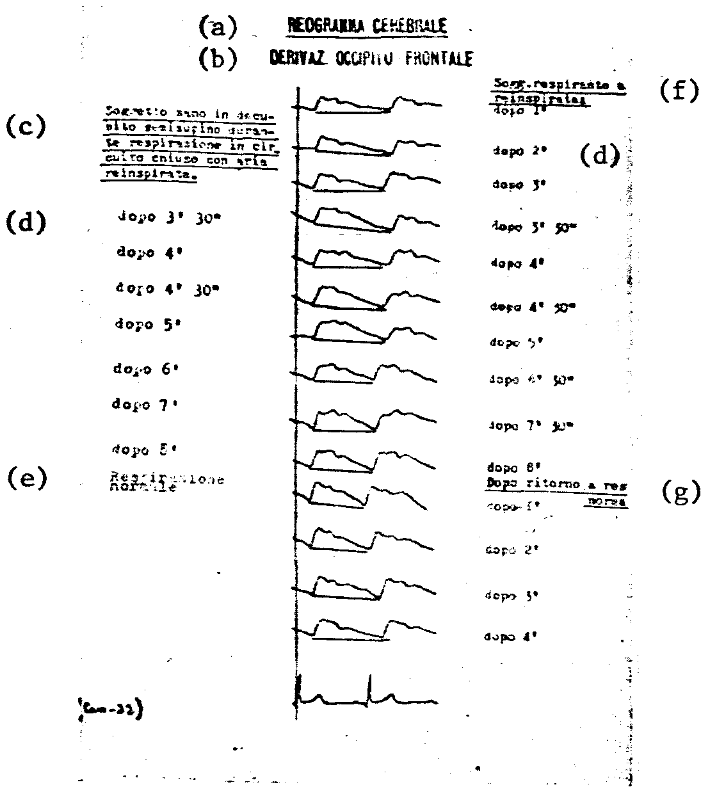

Figure 16

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Healthy subject, semi-reclining, breathing used air in closed circuit; (d)- After; (e)- Normal respiration; (f)- Subject breathing used air; (g)- After return to normal breathing.

are ascertained, i.e., an increase in ascending limb corresponding to a decrease in vascular resistance, and enlargement of the area under the curve corresponding to increased blood flow.

We now present the cerebral rheogram of the same patients of Figures 14 and 16 subjected to respiration of pure oxygen using equipment with intermittent positive pressure (Figure 17).

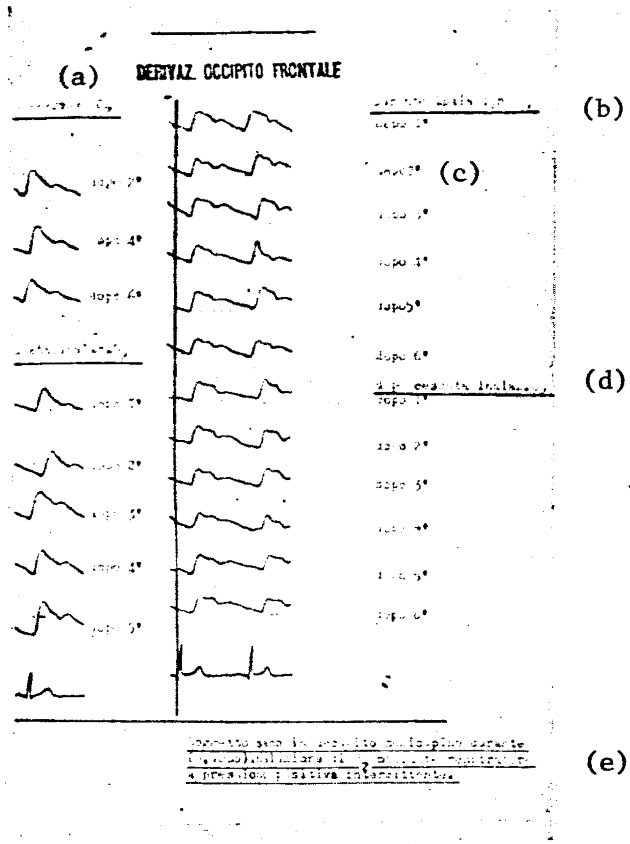

Figure 17

(a)- Occipito-frontal reading; (b)- While inhaling O_2; (c)- After; (d)- After cessation of O_2 inhalation; (e)- Healthy subject, semi-reclining, during (or after) inhalation of O_2 by means of a respirator with positive intermittent pressure.

These tracings show, even though less apparently, variations of an opposite type. In these circumstances, in fact, no apparent flow modifications occur, because the flow - which is subordinated to oxygen consumption - is amply guaranteed. The morphological variations recorded during hyperoxia consist of a reduction in the ascending limb, and this despite the area under the curve. Comparison /220

of the tracings during hyperoxia and hypercapnia in the same subjects shows an opposite situation.

With this first series of experiments conducted on young, healthy subjects, we have come across a correspondence between flow variations and sphygmoplanimetric changes in the rheographic tracing and, in particular, between the ascending limb and the behavior of the cerebral vascular resistance with respect to arterial pressure.

Let us now consider the cerebral rheographic tracing under some pathological conditions. /221

The state of severe anemia, from the cerebral hemodynamic aspect, permits changes similar to those occurring in the state of hypercapnia, i.e., an increased flow of blood because of decreased vascular and cerebral resistances. The lack of oxyhemoglobin - by endangering the metabolic oxygen consumption - is the reason for this compensating reaction.

In the cases which we shall present of severe anemia of different types, we must bear in mind the influence of another factor besides the different pressure, i.e., a possible vascular sclerotic state related to age. The first case (Figure 18) is that of a patient 45 years of age who recovered from severe acute anemia with hematemesis and melena from esophageal varices. Since it was possible to make cerebral /222 rheographic tracings during the anemic state, and after it had been opportunely corrected by transfusions, this case lends itself particularly to comparative considerations. At the moment of the first tracing, the patient showed a hematocrit of 27% and an arterial

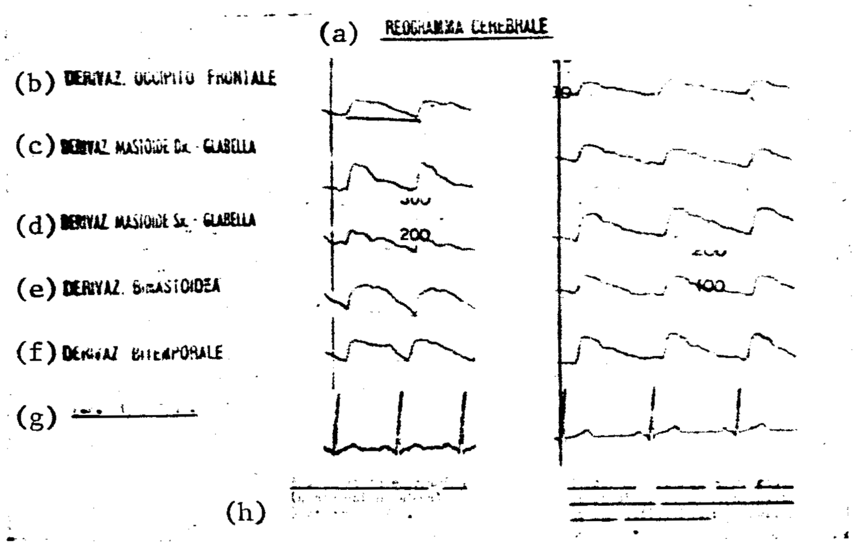

Figure 18

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g, h)- [illegible in original].

pressure of 135/80 mm Hg. The corresponding tracing gives occipito-frontal, bimastoid, and bitemporal readings with a flattened profile, with a decidedly plateau-like peak and moderately reduced area under the curve. The ascending limb displays a height within normal limits. Bearing in mind the pressure value, we may conclude that the flow should re-enter normal limits; the fact that the peak displays a plateau-like outline should induce us to regard this profile as an attempt to increase the area under the curve.

At the second check, the patient had a hematocrit of 40% and an arterial pressure of 100/60 mm Hg. The rheograph outline in the same readings is modified with a peak which is now less plateau-like, a

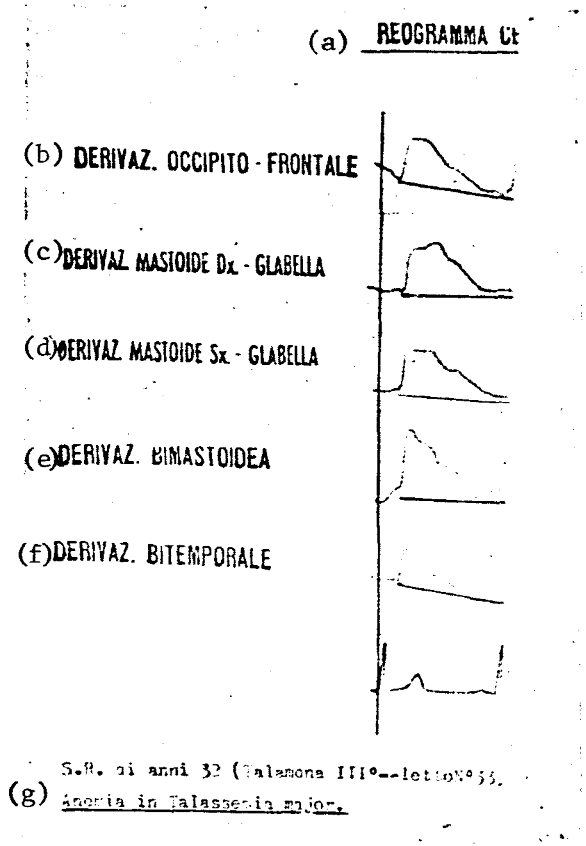

Figure 19

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- S.R., age 32 (Ward 3, Bed 33), anemia in major talassemia.

reduced area under the curve, and lower ascending limb. These details, compared with the preceding, speak clearly in favor of flow reduction by vasoconstriction.

One of our first conclusions could therefore be that, during the /223
anemic state in this patient, the rheographic profile displays a comparative increase in area under the curve with a slight increase in

the ascending limb. Taking account of the pressure value, we find that the flow under this condition is decidedly augmented with respect to that which can be deduced from the second tracing. In this patient, one detail - which leaves us perplexed because it will not enter perfectly into the proposed theoretical system (see page 15) - is the area under the curve and morphology of the second tracing, which we may consider as the expression of the subject's normal conditions. With a pressure of 100/60 mm Hg, we would have expected more area under the curve here.

Let us now consider two other cases of severe anemia. The first case (Figure 19) is that of a 32-year-old patient, arterial pressure 130/70 mm Hg, with severe anemia. The rheographic profile shows a /224
greater than normal area under the curve with a steep and high ascending limb and a rounded peak. The outline, although large, as a whole shows normal morphology; the deductions which we can make are the following: high and steep ascending limb (reduced vascular resistances) and large area under curve (increased flow). These two findings, correlated with normal pressure, can indicate a process of vasodilatation with increased cerebral flow.

The third case is represented by a 63-year-old woman afflicted with severe pernicious anemia (Figure 20), with an arterial pressure of 170/90 mm Hg. The rheographic profile presents the following characteristics: high ascending limb, but with a prolonged rise time; peak within the norm; and enlarged area under the curve. The height of the ascending limb corresponds to decreased vascular resistances,

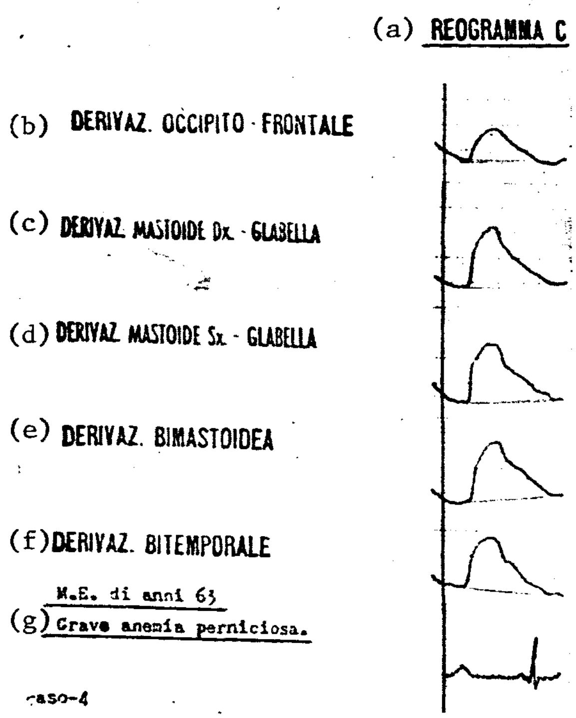

Figure 20

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- M.E., age 63, severe pernicious anemia.

despite the coexistence of decreased vasal elasticity (the rise time /225
expresses the rapidity with which the vessel expands under the arterial pulse). Therefore, taking arterial pressure into account, we can end up with a state of decreased vascular resistances and increased flow in patients with vascular sclerosis. The examination of the fundus confirms this last finding.

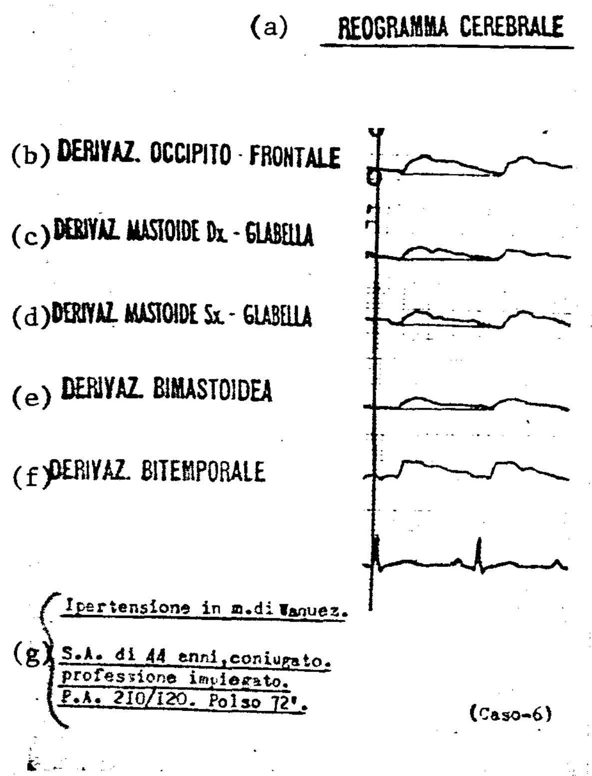

Figure 21

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- Waquez-type hypertension. S. A., 44, male, married, works as employee, arterial pressure 210/120, pulse 72.

In contrast to these cases, we now report an example of polyglobulism (Figure 21). The patient, aged 44, proves to be hypertensive even with pressures of 210/120 mm Hg. An examination of the fundus shows the functional nature of the hypertensive state, with no signs of vascular sclerosis. The rheographic profile presents a clear reduction in area under the curve; the rise time, very evident in the

bitemporal reading, is within normal limits. This tracing presents details which are very distinct from an anemia tracing. The conclusions which we can draw are, therefore, that a clear increase in vascular resistance has occurred and, judging from the pressure, the flow is within normal limits. /226

In this second series of experiments, in which we have considered pathological conditions characterized by a presumable increase in cerebral flow (serious anemias) and reduction in flow (polyglobulism), we believe we have confirmed the correspondence between the behavior of the vascular resistances and the ascending limb. The cerebral flux can be inferred by merely bearing in mind the arterial pressure, because planimetry (of the area under the curve) by itself could lead to erroneous conclusions (see the example of polyglobulism).

Let us now consider several states characterized by an increase in vascular resistances - an increase which we can regard as primitive from an etiopathogenetic standpoint, and not as a reaction compensating for flow variations. These states are represented by essential hypertension and arteriosclerotic hypertension. Arteriosclerosis with normal arterial pressure must be regarded as a condition in which the cerebral vascular resistances are not increased. From Kety's research it follows, in fact, that normotensive arteriosclerosis patients display cerebral vascular resistance values which are moderately elevated and a flow which is more or less reduced, with a cerebral O_2 consumption which tends to keep within normal limits.

In Figure 22, we give the cerebral rheogram of an 85-year-old

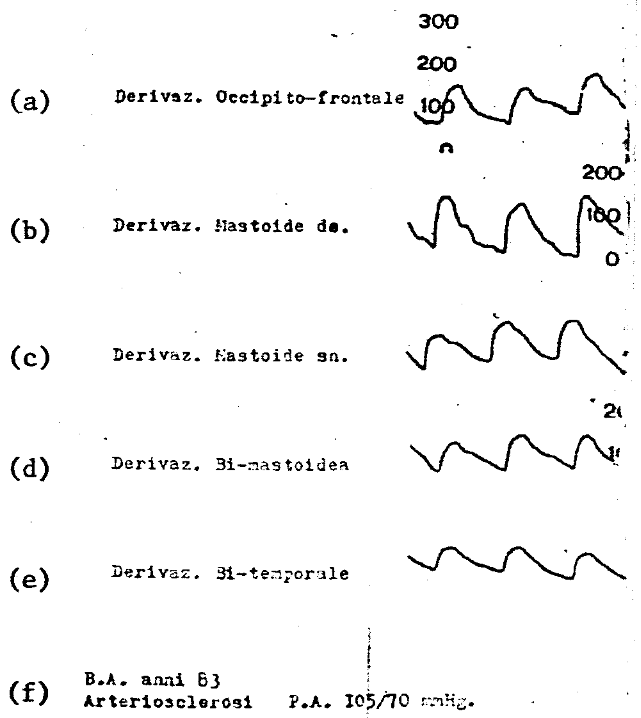

Figure 22

(a)- Occipito-frontal reading; (b)- Right mastoid-glabella reading; (c)- Left mastoid-glabella reading; (d)- Bimastoid reading; (e)- Bitemporal reading; (f)- B.A., age 83, arteriosclerosis, arterial pressure 105/70 mm Hg.

patient with pressure values of 105/70 mm Hg and fundus findings indicating an apparent non-hypertensive vascular sclerosis. The rheographic profile shows an ascending limb with an increased rise time and an area under the curve beyond normal limits. The morphological difference between the right and left mastoid reading reveals the probable existence of a latent state of localized vasculopathy. The deductions which we can make from this tracing point to a condition of vascular resistances

which have not increased, notwithstanding the evident signs of arterio-sclerosis confirmed by the prolonged rise time. The correlation between arterial pressures which are decidedly below normal and high ascending limb leads us to believe that the flow is within normal limits.

Let us now consider a case of arteriosclerotic hypertension. The tracing in Figure 23 belongs to a female patient 63 years of age with a pressure value of 200/110 mm Hg and findings of apparent vascular sclerosis from an examination of the fundus. This tracing displays in all readings a marked prolongation of rise time with a rather pointed peak and reduced area under the curve. These three elements, linked with the arterial pressure, confirm a state of hypertensive vascular sclerosis with a flow which can be kept within normal limits (compare Figures 22 and 23).

In Figure 24, we give another case of severe arteriosclerotic /227
hypertension in which, in contrast to the preceding, the hypothesis can be advanced that there is a coexistent reduction in cerebral flow. In fact, in this case, although hypertensive values like the preceding ones are taken, both the morphological signs of vascular sclerosis (rise time apparently prolonged) and the marked reduction in area under the curve must cause us to assume a state of reduced flow. From a clinical point of view, in fact, this patient presented apparent symptoms of impaired cerebral function, of the demential senilis type. Figure 25 adduces another case of hypertension of renal character. The 68-year-old patient, with a pressure of 210/140 mm Hg and signs of marked hypertensive vascular sclerosis with hemorrhagic foci from an

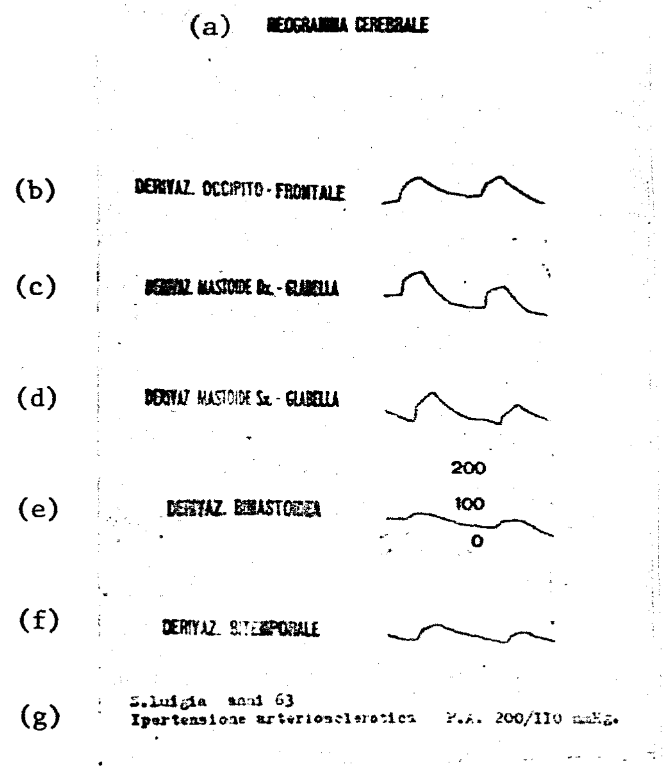

Figure 23

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- S. Luigia, age 63, arteriosclerotic hypertension, arterial pressure 200/110 mm Hg.

examination of the fundus, has an azotemia value of 0.90 mg %. It can be deduced from the rheogram that the flow is kept within normal limits /228 or even superior to them. In fact, a comparison of the pressure value with the height of the ascending limb and the size of the area under the curve leads us to predict a higher-than-normal flow. One possible explanation could be that there is a condition of incipient hemorrhage

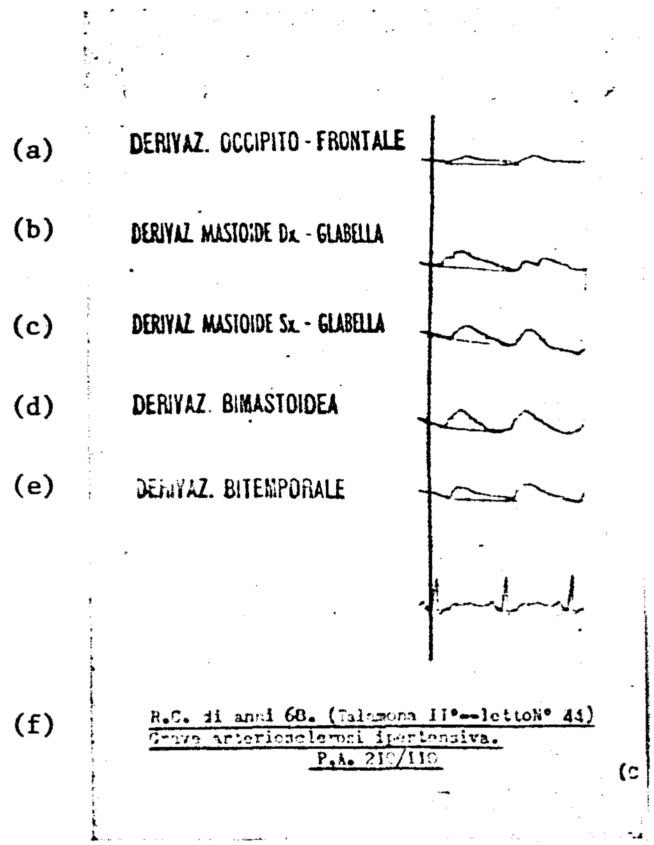

Figure 24

(a)- Occipito-frontal reading; (b)- Right mastoid-glabella reading; (c)- Left mastoid-glabella reading; (d)- Bimastoid reading; (e)- Bitemporal reading; (f)- R.C., age 68 (Ward 2, Bed 44). Severe hypertensive arteriosclerosis. Arterial pressure 210/110.

or an increased flow as the reaction to the hyperazotemic state.

Let us continue our considerations with a series of rheograms made on hypertensive patients treated with Alphamethyldopa. Figure 26 records the rheographic tracings made before and during therapy on a 34-year-old patient with essential hypertension. Arterial pressure was initially 190/130 mm Hg. The corresponding rheogram displays a distinct reduction

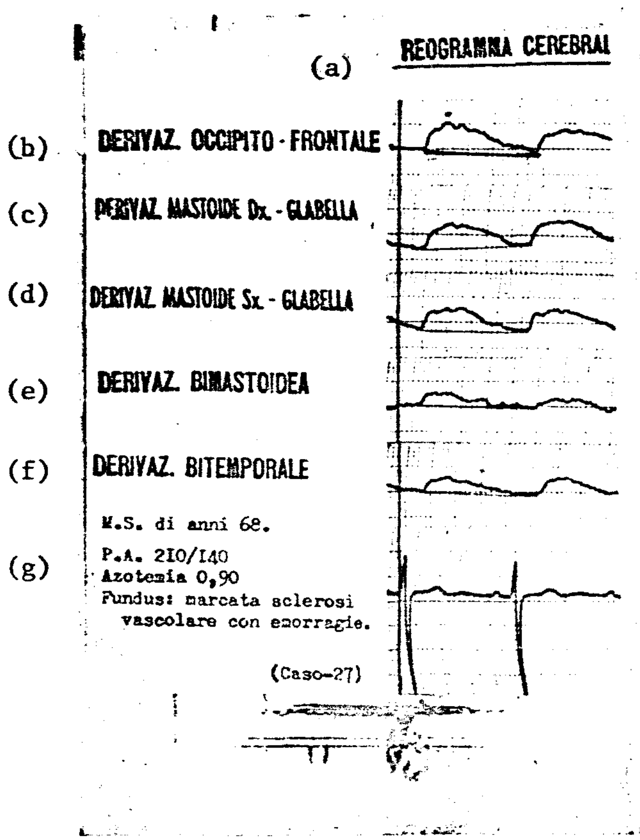

Figure 25

(a)- Cerebral rheogram; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- M.S., age 68, arterial pressure 210/140, azotemia 0.90, fundus -- marked vascolar sclerosis with hemorrage.

in area under the curve, with morphological characteristics favoring an increase in vascular resistences. The tracing made after 24 hours, when the arterial pressure had already fallen to values of 165/120 /229
mm Hg, shows a clear change in the rheogram, which now presents a higher ascending limb and a markedly-enlarged area under the curve. The third check five days later, when pressure had then been stabilized

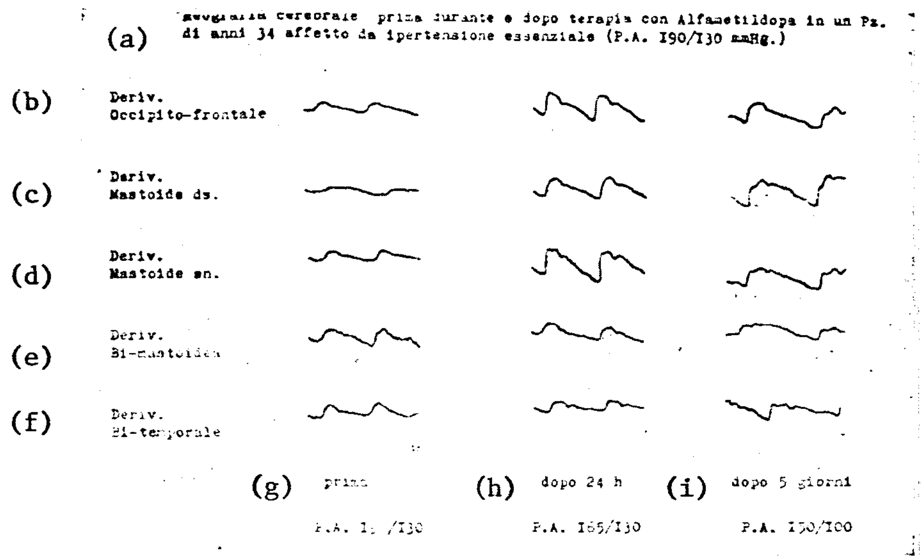

Figure 26

(a)- Cerebral rheogram before, during, and after Alphamethyldopa therapy in a 34-year-old patient with essential hypertension (arterial pressure 190/130 mm Hg); (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- before; (h)- after; (i)- after 5 days.

at values of 150/100 mm Hg, reveals a still different profile characterized by an ascending limb of normal height, with a decidedly-broadened peak and a normal area under the curve. A comparison of these three shows, above all, the possibility of assuming that the variations induced in the cerebral region result from a correction of a hypertensive state. There is a clear relation between the drop in arterial pressure and the increase in ascending limb and area under the curve. Based on this comparison and bearing in mind the relations between pressure and area under the curve, we can advance inductive

hypotheses in the sense of a reduction in vascular resistances accompanied by a normalization of the cerebral flow. /230

The following case (Figure 27*) concerns the same experiment conducted on a 39-year-old patient with chronic hypertensive and hyperazotemic nephritis. In this case, unlike the preceding, examination of the fundus gave evident signs of vascular involvement. The three rheographic profiles lend themselves to similar considerations. Here, too, we have obtained a successive drop in arterial pressure and here, too, we find a progressive increase in the area under the curve by an increase of ascending limb. These reactions are less evident than in the preceding case, notwithstanding that the drop in pressure values was the same. In this case, the coexistence of vascular sclerosis limits the reduction of the vascular resistances. Insofar as the flow is concerned and if the pressure is taken into /231
account, the deductions should lead us to assume that a state of apparent cerebral flux reduction was established. In the same patient, after evaluating the response to a rapid hypotensive effect, we repeated the experiment of intravenous injection of Alphamethyldopa.

In Figure 28, we give the occipito-frontal rheogram taken at intervals in the 35 min. following intravenous injection. The variations in the rheogram clearly indicate that there is a prompt and progressive cerebral vasomotor reaction resulting from the drop in general arterial pressure.

---

* Note: Figures 27 and 28 are missing in the original foreign text.

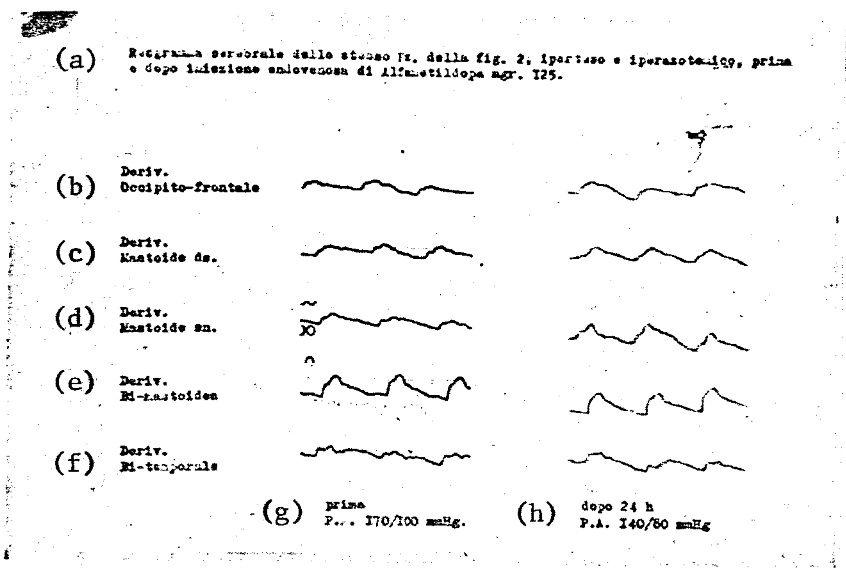

Figure 29

(a)- Cerebral rheogram of same patient as in Figure 2, hypertensive and hyperazotemic, before and after intravenous injection of 125 mg of Alfamethyldopa; (b)- Occipito-frontal reading; (c)- Right mastoid-glabella reading; (d)- Left mastoid-glabella reading; (e)- Bimastoid reading; (f)- Bitemporal reading; (g)- Before, arterial pressure 170/100 mm Hg; (h)- After 24 hours, arterial pressure 140/80 mm Hg.

In Figure 29, we present complete rheograms before and 24 hours after the experiment. All these details confirm the possibility of using cerebral rheography to take the prompt reactions which occur in the cerebral region as a result of administering drugs with clear hypotensive effect.

Conclusions

The interpretation of cerebral rheography is a problem which is at present the subject of discussion because, notwithstanding the numerous findings which have been made, cerebral rheography often appears to be

arbitrary and variable in clinical practice. The propositions formulated between variations in electrical conductivity and manners of /233 blood flow appear convincing and realistic in many cases, and in many others the same propositions are no longer possible. From attentive observation of the rheographic tracings compared with the clinical findings, we realized that another factor appeared to be decisive for interpretative purposes, i.e., the value of arterial pressure. With this remark in mind and reviewing the theoretical assumptions, we can define and document the inexactness of the assumption that establishes a proportional relationship between electrical conductivity and volume, and therefore blood flow, in the region under examination. In the laws of hemodynamics, indeed, the relationship between variations in volume and flux is dependent on pressure; just as with the volume of a vessel, when pressure becomes greater, flow increases.

The cerebral rheogram can, therefore, be interpreted as the final expression of the interaction of diverse factors, i.e., flow, resistances, and pressure. The task which we set ourselves was therefore that of ascertaining what relationship existed between the individual parts of the rheographic outline and each one of these factors. Preceding works had already shown the relationship between behavior of the ascending limb and the anatomicophysio-pathological character of the vessels. One correlation, which is minor insofar as it is not constant, is, on the other hand, that between flow and area under the curve. From our clinical investigations conducted for this very purpose, we have /234 confirmed the correlation between the ascending limb of the rheographic

outline and behavior of the vessels, and have defined the relationship between flow and area under the curve as the result of pressure and resistance.

With this interpretative criterion, we have illustrated a series of clinical cases predominantly characterized by alterations in cerebral flow, vascular resistance, or pressure, and by using the concepts of cerebral hemodynamics - which have already become well-known in the literature - we have been able time and time again to establish a clear parallelism between clinical details and cerebral rheogram. Of course, the deductions cannot be made absolutely, but only relatively, because rheographic investigation reflects the variations of a phenomenon and does not define it quantitatively.

The conclusions at which we can arrive as the result of our present experience are, above all, concretely expressed in the main significance which can be attributed to rheography, as the expression of the modes of the blood flow in the cerebral region as a function of the vascular resistance and arterial pressure.

Such an investigation can make valid contributions to diagnostic problems, only if supported by the knowledge of these last factors and of the manifold variants which characterize every individual clinical case. This is, moreover, also true of all the instrumental investigations which are presently accompanying the interpretative evolution of medical diagnosis, which contributes to a knowledge of the disease process manifestations which could not otherwise be known.

REFERENCES

1. Aird, R. B. The Retinal Circulation as an Index of Cerebral Circulation from the Viewpoint of the Neurologist. Angiology, 193, 1949. /235

2. Bovet, D. The Present State of the Pharmacodynamics of Cerebral Circulation (Stato attuale della farmacodinamica del circolo cerebrale). Il Triangolo, Vol. V, 1961.

3. Bach, L. M. N. The Organization of Vasomotor Control by the Central Nervous System. Am. Heart. J., 55, 323, 1958.

4. Bernsmeier, A., Gottstein, U. Cerebral Blood Supply and Age (Irrorazione cerebrale e detà). Verh. Dtsch. Ges. Kreislaufforschg., 24, 248, 1958. /236

5. Best, C., Taylor, N. B. The Physiological Basis of Medical Practice. William & Wilkins Co., Baltimore, 1945.

6. Bouckaerty, J. J., Heymans, C. On the Reflex Regulation of the Cerebral Blood Flow and the Cerebral Vasomotor Tone. J. Physiol., 84, 67, 1935.

7. Belluschi, A., Bottoni, A. Atti Acc. Med. Lomb., XV, 874-883, 1960; Annales d'Ocul., 194, 320-327, 1961.

8. Belluschi, A., Vacchini, V., Vecchio, C., Panzacchi, G. Evaluating Cerebral Vascular Resistance in Emphysematous Patients (Valutazione delle resistenze vascolari cerebrali nei pazienti enfisematosi). L'Ospedale Maggiore, 9, 1963.

9. Belluschi, A., Vacchini, V. Study of the Correlation Between pH, Hematic CO_2, and Cerebral Vascular Resistance in Emphysema Patients (Studio sulla correlazione tra pH, CO_2 ematico e resistenze vascolari cerebrali negli enfisematosi). L'Ospedale Maggiore, 9, 1963.

10. Belluschi, A., Vacchini, V. Clinical Contributions of Ophthalmo-angiodynamometry in the Study of Alterations in Cerebral Hemodynamics (Contributi clinici dell'oftalmoangiodinamometria nello studio delle alterazioni dell'emodinamica cerebrale).

11. Condorelli, L. Physiopathology of the Venous Circulation (Fisiopatoligia della circolazione venosa). Catania, 1947.

12. Davis, L., Perret, G. Brit. Surg., 34, 307, 1947.

13. Della Torre, L. Sphygmography (La sphygmographie). Ed. Accad. Pontificia, Rome, 1946.

14. De Vecchi, B., Rogers, W. N. Report on Arterisclerosis (Relazione sulla arteriosclerosi). Cuore Circol., 19, 236, 1935.

15. Donato, L. Metodology for Using Radio-Isotopes in Circulatory Diagnostics (Metodologia per l'impiego dei radioisotopi nella diagnostica circolatoria). Min. Med., 46, 50, 1955.

16. Douzelot, et al. Study of Cerebral Circulation by Transcranial Diagraphy (L'étude de la circulation cérébrale par la diagraphie transcranienne). Arch. Mal. Coeur, 44, 219, 1955.

17. De Langen, C. D. Blood Pressure, Pulse Pressure, and Tissue Flow. Cardiologia, 33, 249, 1958.

18. Dewar, H. A., Davison, L. A. G. The Cerebral Blood Flow in Mitral Stenosis and Its Response to Carbon Dioxide. British Heart Journal, 20, 516, 1958.

19. Beccatini, U., Cangi, G., Della Corte, M. Modern Criteria in Interpretation of Rheography (Moderni criteri nella interpretazione della reografia). Riforma Medica, 76, 1962.

20. Facchini, G., Garbini, G. C. Prospects of Early Diagnosis of Cerebral Arteriosclerosis (Prospettive di diagnosi precoce dell'arteriosclerosi cerebrale). Bull. Sci. Med., 1957.

21. Facci, M., Garbini, G. C. The Normal Rheocardiographic Curve (La curva reocardiografica normale). Min. Cardio-ang., 1957.

22. Forbes, H. S., Wolf, H. G. Cerebral Circulation: Vasomotor Control of Cerebral Vessels. Arch. Neur. Psych., 19, 1057, 1948.

23. Frau, G. Recording Skin Temperature in Studying Peripheral Vasculopathies (La registrazione della temperatura cutanea nello studio delle vasculopatie periferiche). Fol. Cardiol., 6, 215, 1947.

24. Furbetta, D., Bufalari, A., Santucci, F. Research on Rheocardiography: the Esophageal Rheocardiogram (Ricerche in tema di recocardiografia: il reocardiogramma esofageo). Fol. Cardiol., 13, 281, 1954.

25. Feruglio, F. S. Omnia Medica, 32, 337, 1954.

26. Forbes, H. S. Physiologic Regulation of the Cerebral Circulation. Arch. Neurol. and Psychiat., 43, 804, 1940.

27. Garbini, G. C., Posteli, T., and Montali, M. Cerebral Sphygmic Rheographic Notes (Rilievi sfigmici reogarafici cerebrali). Riv. Med. Bologna, 3, 435, 1957.

28. Gigli, G., Donato, L., Bartolomei, G., and Bianchi, R. The Use of Radio-Isotopes in Studying Hemodynamics and the Importance of New Methodologies in Cardiocirculatory Symptomatology (L'impiego dei radioisotopi nello studio dell'emodinamica e importanza delle nuove metodologic nella semeiologia cardio-circolatoria). Min. Nucleare, 1, 1, 1957.

29. Goez, R. H. Clinical Pletysmography. South Africa M. J., 22, 391, 1948.

30. Gentilli, R. Use of Radio-Isotopes in Studying Certain Problems Connected with Blood Supply (L'impiego di radioisotopi nello studio di alcuni problemi connessi alla rirrolazione sanguigna). Min. Card. ang., IV, 272, 1956.

31. Holzer, W., et al. Rheocardiography (Rhéocardiographie). Maudrich, Wien, 1945.

32. Honig, C. R., Tenney, S. M. Determinants of the Circulatory Response to Hypoxia and Hypercapnia. Am. Heart J., 53, 687, 1957.

33. Hansel, F. K. Vascular Headaches and Related Phenomena. Angiology, 7, 457, 1956.

34. Kaindl, F. Rheography of Peripheral Arteries (Rheographie peripher Arterien). Arch. Kreisl Forsch, 20, 247, 1954.

35. Kety, S. S., Schmidt, C. F. Nitrous Oxide Method for Quantitative Determination of Cerebral Blood Flow in Man: Theory Procedure and Normal Values. J. Clin. Invest., 27, 476, 1948.

36. Kety, S. S., Schmidt, C. F. The Effects of Active and Passive /237
Hyperventilation on Cerebral Blood Flow, Cerebral Oxygen

Consumption, Cardiac Output and Blood Pressure of Normal Young Men. J. Clin. Invest., 25, 107, 1946.

37. Kappert, A. Clinical Plethysmography (Klinische Plethyamographie). Cardiologla, 24, 353, 1954.

38. Kety, S. S., et al. J. Clin. Invest., 27, 484, 1948.

39. Kety, S. S., et al. J. Clin. Invest., 27, 511, 1948.

40. Kety, S. S., et al. J. Cron. Dis., 3, 478, 1956.

41. Kety, S. S., et al. J. Clin. Invest., 27, 500, 1948.

42. Kety, S. S., et al. Cerebral Vascular Disease. J. Wright and H. Luckey, New York.

43. Malamani, V. and Galeazzi, L. Rheocardiography in Physiopathological Cardiac and Circulatory Evaluation (La reocardiografia nella valutazione fisiopatologica del cuore e della circolazione). Boll. Soc. Med. Chir., Pavia, No. 1-2, 1949.

44. Malamani, V. and Brangi, G. P. Pharmacological Variations in the Rheogram of the... (Variazioni farmacologiche del reogramma del...).*

45. Malamani, V. and Brangi, G. P. Pharmacological Variations in the Rheogram of the Cranium and Hepatic Region (Variazioni farmacologiche del reogramma del cranio e della regione epatica). Fol. Cardiol, 14, 381, 1955.

46. Malamani, V., Bobba, P., and Storti, D. Effects of Hypoxia on Rheograms of the Cranial Articulations and the Hepatic Region (Gli effetti della ipossia sui reogrammi degli arti. del cranio

---

* Note: Reference incomplete in the original foreign text.

e della regione epatica). Atti XVIII Congr. S.I.C. Trieste, page 300, 1956.

47. Merlen, J. F., Vanrapenbusch, R., and Cachera, J. P. Ballistograms and Rheograms from the Viewpoint of Theoretical Hemodynamics (Le balistogramme et le rhéogramme vus sous l'angle de l'émodynamique théorique). Arch. Mal. Coeur., 46, 1038, 1953.

48. Merlen, J. F., and Bozal Urzay, V. Ballistograms and Rheograms from the Viewpoint of Theoretical Hemodynamics (El balistograma y el rheograma vistos bajo el angulo de la hemodinamica teorica). Rev. Esp. Cardiol., 8, 15, 1954.

49. Merlen, J. F., Vanrapenbusch, R., and Cachera, J. P. Rheocardiography: a Critical Study (La rhéocardiographie étude critique). Presse Med., 62, 1251, 1954.

50. Merlen, J. F. Exploring the Cerebral Arteries by Rheography (Exploracion de las arterias cerebrales por la reografia). Angiologia, 7, 298, 1955.

51. Margaria, R., De Caro, L. Principles of Human Physiology (Principi di fisiologia umana). Ediz. Vallardi.

52. Mori, K. Cerebral Circulation and Metabolism in Brain Stem Injury. Japanese Circulation Journal, 24, 397, 1960.

53. Morello, A., Bartecek, A., Stellar, S., and Cooper, I. S. Angiographic Evaluation of Vasodilatation in Cerebral Vessels. Angiology, 7, 16, 1956.

54. Munch, O., Lassen, N. A. Bilateral Cerebral Blood Flow and Oxygen Consumption in Man by Use of Krypton 85. Circul. Res., 5, 163, 1957.

55. Murphy, J. P. Recent Advances in the Investigation and Management of Cerebrovascular Disease. Circulation, 13, 281, 1956.

56. Nyboer, J. D., Bagno, A. Barnett, and Helsey, R. Radiocardiograms: Electrical Impedance Changes of the Heart in Relation to Electrocardiograms and Heart Sounds. J. Clin. Invest., 19, 963, 1940.

57. Nyboer, J. D. Electrical Impedance Plethysmography. Circulation, 2, 811, 1950.

58. Nyboer, J., Hreider, M. M., and Kannapel, L. Quantitative Studies of Electrical Conductivity of the Peripheral Body Segments: Basic and Practical Considerations. Ann. West. M. a. S., 5, 1, 11, 1951.

59. Nyboer, J. and Watson, T. R. Jr. Constant Mass Displacement Ballistocardiography and Electrical Impedance Plethysmography. J. Lab. Clin. Med., 46, 270, 1955.

60. Orlandi, G., Garbini, G. C., and Gentili, G. Possibilities of Rheographical Study of Collateral Compensatory Circles in Cerebral Vasculopathies (Possibilità di studio dei circoli collaterali di compenso nelle vasculopatie cerebrali mediante metodo reografico). Communicazione Congr. Soc. Ital. Angiol. Stresa, April 22, 1957.

61. Pratesi, F., Nuti, A., Sciagrà, A. Cranial Rheography. Functional Tests in Diagnosing Cerebral Vasculopathies (Reografia cranica. Prove funzionali nella diagnostica delle vasculopatie cerebrali). Min. Med., 48, 1957.

62. Piegari, V., Purini, T. First Observations on Rheography in Venous Stasis by Studying the Peripheral Vascular Tone (Prime osservazioni

sulla reografia in stasi venosa per lo studio del tono vascolare periferico). Min. Cardioang., 11, 224, 1963.

63. Purini, T., Piegari, V. Experimental Research on the Postural Response of Rheography (Ricerche sperimentali per lo studio della risposta posturale reografica). Min. Cardioang., 11, 225, 1963.

64. Perego, M. A. The Symptomatological Value of Rheography in Studying Peripheral Vasculopathies (Il valore semeiologico della reografia nello studio delle vasculopatie periferiche). Rass. Fis. Clin. Terap., 30, 10, 1958.

65. Perego, M. A., Patrigniani, S., Allori, L., Sonnino, S. Functional Symptomatology of Peripheral Vasculopathies: Postural, Exertional, and Pharmacological Modifications, Particularly in Regard to Rheograms (Semeiologia funzionale delle vasculopatie periferiche: modificazioni posturali, da sforzo e farmacologiche particolarmente in rapporto al reogramma). Ras. Fis. Clin. Terap., 30, 11, 1958.

66. Perego, M. A., et al. Rheography in the Study of Regional Venous Hemodynamics (La reografia nello studio dell'emodinamica venosa distrettuale). Policlinico Sez. Pr., 67, 38, 1960.

67. Perego, M. A., et al. Cranial Rheography in Certain Syndromes of Changed Endocranial Circulation and Its Pharmacological Modifications (Il reogramma cranico in alcune sindromi di alterata circolazione endocranica e sue modificazioni farmacologiche). Ras. Fis. Clin. Terap., 30, 9, 1958. /238

68. Perego, M. A., et al. Report, September 1960, to the Soc. It. Geron. e Ger. (Relaz. settembre 1960 alla Soc. It. Geron. e Ger.)*

* Note: Reference incomplete in the original foreign text.

69. Polzer, K., Shuhfried, H. Rheography, a New Method of Checking Blood Flow (Reografia, un nuovo metodo per il controllo del flusso sanguigno). Min. Cardioang., 4, 369, 1956.

70. Pratesi, F., et al. Cranial Rheography; Orientational Clinical Observations (Reografia cranica; osservazioni cliniche orientative). Fol. Angiol., 1957.

71. Proceedings of the First Symposium on the Physiopathology of Cephalic Circulation (Relazione atti I° Simposio di fisiopatologia del circolo cefalico). Florence, March, 1958.

72. Serra, P. Examination of Cerebral Circulation by the Rheographic Method (L'esame della circolazione cerebrale col metodo reografico). Ann. Ital. Chir., 32, 76, 1955.

73. Sicuteri, F., et al. Sett. Med., 45, 1, 46, 1957.

74. Samuel, S. S. Vascular Diseases (Malattie vascolari). S. E. U., Rome, 1958.

75. Iandolo, C., Puddu, V. Syncope; Physiology and Clinical Care of Acute Cerebral Anoxia (La sincope; fisicatologia e clinica della anossia cerebrale acuta). Pozzi editore, Rome.

76. Whiteleather, J., Keirns, M. M. Transactions of the American College of Cardiology. 6, 135, 1956.

77. Weigelin, E. Ber. Dtsch. Ophthal. Ges., 54, 50-55, 1949.

78. Weigelin, E. Ber. Dtsch. Ophthal. Ges., 55, 227-232, 1949.

79. Weigelin, E. Ber. Dtsch. Ophthal. Ges., 55, 394-398, 1949.

80. Weigelin, E., Frusberg, O. Ophthalmologica, 119, 292-314, 1950.

81. Weigelin, E., et al. Dynamometric Studies on the Relations Between

Cerebral Disorders and Brain Circulation in Hypertensive. Graefes Arch. Ophthal., 161, 601-14, 1960.

82. Weigelin, E., Niesel, P. Retinal Artery Pressure in Change of Bodily Position (Der netzhautarteriendruk bei Anderung der Korperlage). Graefes Arch. Ophthal., 150, 374-384, 1950.

Scientific Translation Service
4849 Tocaloma Lane
La Canada, California